

## Patterns of Dental Enamel Defects at Ancient Mendes, Egypt

NANCY C. LOVELL\* AND IRA WHYTE  
*Department of Anthropology, University of Alberta,  
Edmonton, Alberta T6G 2H4, Canada*

**KEY WORDS** dental pathology; systemic stress; malnutrition;  
enamel hypoplasia

**ABSTRACT** The dental remains of 88 individuals from Old Kingdom, First Intermediate, and Greco-Roman periods at the ancient Egyptian site of Mendes (Tell er-Rub'a) were examined for dental enamel hypoplasia, and the results reported here provide some of the first comparative data on enamel defects in ancient Egypt. Overall, 48% of the individuals in the sample have one or more teeth with hypoplasia, with 17% of permanent teeth and 8% of deciduous teeth affected. The permanent teeth account for 87% of the total number of affected teeth, a prevalence over deciduous teeth that is significant at  $\alpha = 0.05$ . Permanent and deciduous teeth display different patterns of hypoplasia, with the former exhibiting both discrete pitting and linear furrowing, and the latter exhibiting only pits. Teeth with linear defects significantly outnumber those with pits by a factor of more than three to one. Only permanent canines display more than one lesion on a tooth, with a mean of 1.4 defects per affected tooth. Although calculation of the age of insult from lesion position is imprecise, it appears that stress episodes occurred most commonly between approximately 3–5 years of age. The presence of pits in the deciduous dentition, however, suggests that physiological stresses began in utero. There is no statistically significant difference in the frequency of enamel defects between males and females. An observed decrease in the frequency of defects from the Old Kingdom period to the subsequent First Intermediate and Greco-Roman periods is not significant at  $\alpha = 0.05$ , although such a decrease is expected given epigraphic and other data that refer to prolonged drought and malnutrition in the late Old Kingdom. The calculated  $\chi^2$  value of 3.83 is significant at the 0.10 level, however, and since our sample is rather small and the magnitude of the chi-square statistic is a function of sample size, we recommend that future research investigate further the relationship between the frequency of enamel defects and the time period in which they occur. *Am J Phys Anthropol* 110:69–80, 1999. © 1999 Wiley-Liss, Inc.

Developmental defects of dental enamel take two principal forms: enamel hypoplasia, and enamel opacity or hypocalcification. Enamel hypoplasia is a quantitative defect of tooth crown enamel that most commonly appears as discrete pitting or as horizontal furrows (Hillson and Bond, 1997; Nikiforuk and Fraser, 1981; Pindborg, 1970; Seow, 1991) and that is symptomatic of a disruption in ameloblasts' enamel secretion during

crown development. Although the same physiological insults that lead to enamel hypoplasia also may lead to enamel hypocalcification, the latter defect results from dis-

Grant sponsor: Social Sciences and Humanities Research Council of Canada; Grant numbers: 410–91–1162, 410–95–0254.

\*Correspondence to: Nancy C. Lovell, Department of Anthropology, 13–15 Tory Building, University of Alberta, Edmonton, Alberta T6G 2H4, Canada. E-mail: Nancy.Lovell@ualberta.ca

Received 25 February 1997; accepted 4 May 1999.

ruptions during enamel mineralization or maturation, which occurs after the enamel has been deposited. The etiology of the disruption is not always discernible, but enamel hypoplasia appears to be a sensitive reflection of physiological stress (Nikiforuk and Fraser, 1981; Pindborg, 1970; Sarnat and Schour, 1941; Skinner and Goodman, 1992). This stress may have been brought about by hereditary factors, but current evidence indicates that hypoplastic lesions resulting from environmental stressors are more common than those of genetic origin (Goodman and Armelagos, 1985; Goodman and Rose, 1990, 1991; Hillson, 1996; Lukacs, 1992; May et al., 1993; Moggi-Cecchi et al., 1994; Seow, 1991; Skinner and Goodman, 1992). Enamel hypoplasia has been significantly correlated, for example, with social economic status and decreased physical stature (Blakey and Armelagos, 1985; Cook and Buikstra, 1979; Enwonwu, 1973; Goodman et al., 1980). Malnutrition is one environmental stressor that historically was viewed as a primary cause of enamel defects, but the interaction of two or more factors, particularly diet and disease, is now understood to be involved. A recent interpretation of enamel defects suggests that their presence in a skeletal sample demonstrates individuals' ability to adapt biologically to physiological stresses and hence should be interpreted as reflecting resilience, or fitness, rather than frailty (Wood et al., 1992), which is in accord fundamentally with the traditional interpretation of enamel defects as stress markers (e.g., Lukacs, 1994).

Deciduous teeth develop from approximately 3 months in utero until about 1 year after birth, while permanent teeth develop from birth to approximately 12 years. Because they begin to form in utero, defects on deciduous teeth also give an indication of the physical and nutritional state of the mother (Lukacs, 1992; Seow, 1991). Since tooth formation and maturation occur at predictable rates, it has been common practice to estimate the approximate age of the stress occurrence during growth and development from the measured position of macroscopically visible enamel defects on the tooth crown (Buikstra and Ubelaker, 1994; Goodman and Rose, 1990; Moggi-Cecchi et

al., 1994; Rose et al., 1985). Recent research, however, indicates that both the precision and accuracy of these estimates are likely to be poorer than previously thought, and stresses the value of histological examination (Hillson and Bond, 1997).

In this paper we present the results of an analysis of enamel defects in the deciduous and permanent teeth of individuals interred at ancient Mendes (also known as Tell er-Rub'a) in the eastern central delta of Egypt (Fig. 1), in order to assess the impact of changing socioeconomic and physical environments on health status during the 2,000-year habitation of the site. In spite of the lengthy archaeological record at the site (Brewer and Wenke, 1992; Redford et al., 1988), little is yet known of the nutritional status or disease stresses of the local citizens. Furthermore, with the exception of the discussion by Hillson (1979) of structural defects in his review of the links between diet and dental disease, there are few data published on enamel hypoplasia in ancient Egyptian and Nubian populations.

Archaeological evidence indicates that Mendes was first occupied in the late Predynastic and Archaic Periods (ca. 3200–3000 BC), the formative era of the ancient Egyptian civilization, and was a large and prosperous community during the subsequent Old Kingdom, or Pyramid Age (Brewer and Wenke, 1992; Redford et al., 1988). For most of its history, Mendes was a provincial capital in northern Egypt, perhaps due to its prime location on a major branch of the Nile, where it served as a trade center and connecting point, via the river to Upper Egypt and Nubia in the south, and via the Mediterranean Sea and coastal routes to Palestine and western Asia (Brewer and Wenke, 1992; Wenke, 1991). Although a desert region today, in antiquity the area around Mendes was well-watered by tributaries of the Nile and supported an abundance of plant and animal life. Several areas of the site have been systematically excavated since 1964: the Dynasty XXVI temple precinct (constructed ca. 550 BC); residential and industrial areas which date from the New Kingdom (ca. 1570–1070 BC) through Greco-Roman (ca. 332 BC–AD 395) periods; Predynastic, Archaic, and Old Kingdom cem-

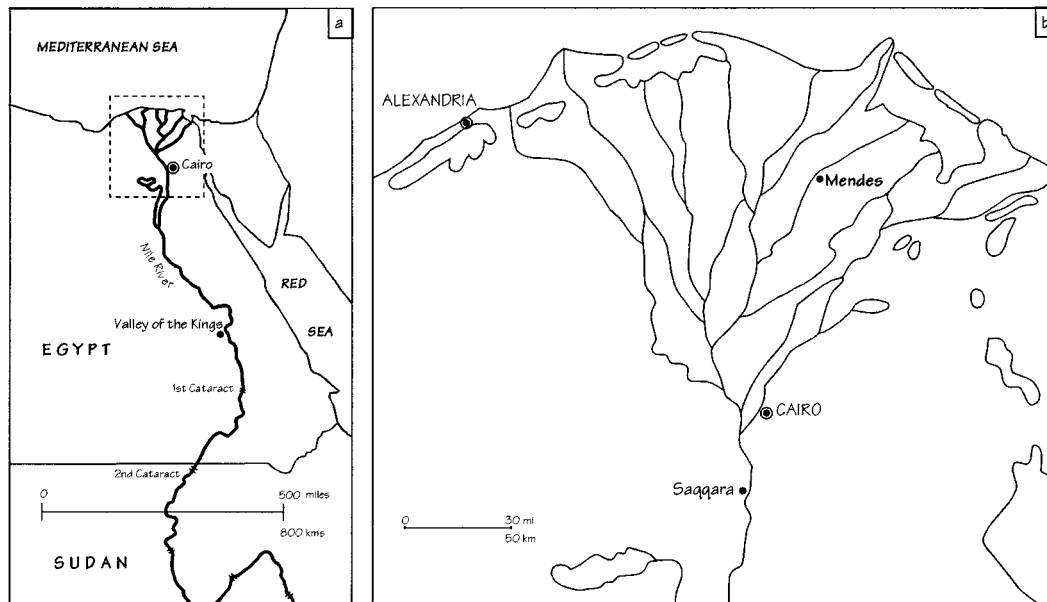


Fig. 1. Maps of the Nile River valley (a) and delta (b), showing sites referred to in the text.

eteries and settlements (ca. 3200–2180 BC); and the satellite cemetery mound of Kom el-Adhem, which dates to the Greco-Roman period (Brewer and Wenke, 1992; Hansen, 1967; Holtz et al., 1980; Lovell, 1992; Wilson, 1982). The area now covered by the site is approximately 85 hectares.

## MATERIALS AND METHODS

### The dental sample

The skeletal remains were recovered at Mendes during sporadic excavations at the site over the past three decades (Brewer and Wenke, 1992; Hansen, 1967; Lovell, 1992) and represent three periods of occupation: the Old Kingdom (ca. 3000–2180 BC), the First Intermediate period (ca. 2180–2040 BC), and the Greco-Roman period (ca. 332 BC–AD 395). The total sample consists of 96 dentitions from 88 individuals (Table 1): 72 permanent dentitions and 24 deciduous dentitions; mixed dentitions appeared in 8 individuals. The sex of each adult was determined by standard osteological methods (e.g., Bass, 1989; Buikstra and Ubelaker, 1994; White, 1991), with emphasis given to sexually dimorphic features of the os coxa. Unfortunately, skeletal remains from the

TABLE 1. Mendes dental sample<sup>1</sup>

	Male		Female		Unknown		Total
	n	%	n	%	n	%	
Permanent dentitions	23	32	26	36	23	32	72
Deciduous dentitions	2	8	0	0	22	92	24
Total	25	26	26	27	45	47	96

<sup>1</sup> Although the total number of observable dentitions as shown here is 96, only 88 individuals comprise the sample analyzed, since 8 individuals possessed mixed dentitions. n, number of individuals; %, [number of individuals of that sex/total number of individuals in that dental sample] × 100. The result is rounded to the nearest percent.

site are quite friable due to the burial conditions (described below), and although apparently well-preserved when brushed free of sand they were often reduced to fragments upon handling. The probable sex of the in situ remains fortunately was determined easily from preserved morphological features of the pelvis. Most of the individuals for whom sex could not be attributed were not examined at the site but were shipped to the senior author for analysis in Canada some 20 years after excavation; regrettably, they did not stand up well to the rigors of excavation, curation, and transport. Teeth, in particular, were often very highly fragmented, and the remains of 49 individuals (in addition to the 88 examined in this

study) did not possess anterior teeth that were preserved adequately for an assessment of enamel defects.

Although the representativeness of the cemetery sample in terms of the populace at Mendes is difficult to determine, we have not detected any bias in burial practices among the nonelite inhabitants of the community, and thus we consider it reasonable to assume that our sample is characteristic of the Mendesian "middle class" (details of burials and their grave accompaniments and discussion of status can be found in Lovell, 1992). Royal or otherwise elite members of Mendesian society were accorded sumptuous burial, as demonstrated by the excavation of three such elite tombs which were looted in antiquity (Hansen, 1967). While other such tombs may remain intact and undiscovered, several episodes of destruction of tombs, sacred animal necropoli, and buildings of a temple complex (Magee et al., 1996; Redford 1993a,b; Redford et al., 1988) suggest that the remains of higher-status individuals may be difficult to find at this site.

### Scoring methods

Data collection procedures were based on criteria outlined by Buikstra and Ubelaker (1994), and data are reported using both the tooth count (number of affected teeth as a proportion of the number of observable teeth) and the individual count (number of affected individuals as a proportion of the number of observable individuals) methods. While the individual count method gives a sense of the proportion of the populace that experienced episodic stress, tooth count frequencies permit a broader comparison among subsamples and/or with other study samples. The 12 anterior teeth of the permanent dentition, considered the most susceptible to stress and therefore the best indicators of enamel defects (Goodman and Armelagos, 1985; Goodman and Rose, 1990; Moggi-Cecchi et al., 1994), and the 20 teeth of the full deciduous dentition were examined. This selection of multiple tooth classes for scoring is recommended over the choice of a single tooth, due to variable susceptibility to developmental surface defects within and between tooth types (Condon and Rose, 1992).

Although we have treated antimeres as separate data points in order to enlarge our tooth count sample sizes and maximize the recovery of information, the tooth count frequencies are calculated relative to the total number of teeth preserved and thus should not affect substantially the true expression of enamel defects in the sample.

Teeth are often the best-preserved skeletal component in an archeological context, but the small size of this study sample can be explained by the taphonomy of the region: contrary to the popular belief that organic preservation in Egypt is uniformly excellent, in this part of northern Egypt it is relatively poor. The delta is now, and has been for millennia, one of the wettest regions in Egypt; yet it is also classified as a hot desert, and the alternating wet and dry conditions are hard on organic remains. Deterioration is further hastened by the evaporative capillary action of moisture from the high water table, which is exacerbated by irrigation. The soil is very alkaline, since there is not enough rainfall to wash away surface salts. In addition, the absence of some single-rooted anterior teeth can be attributed to recovery failure during early excavations and to taphonomic factors (see, e.g., McKeown and Bennett, 1995).

The teeth were assessed visually for hypoplasia and hypocalcifications/discolorations under strong oblique light with the aid of a 10-power hand lens. Regrettably, the conditions governing our excavation and analysis of human remains from Mendes, as established by the Egyptian antiquities authorities, prevent destructive analysis (e.g., thin-sectioning), and therefore we have had to rely solely on macroscopic analysis in this study. While Hillson and Bond (1997) argue convincingly that the age of insult can be accurately determined only by examination of histological defects, an estimate of the *relative* timing of physiological stress during the early, middle, or later years of tooth development can be obtained from the measured position of the macroscopically visible defect on the tooth crown. The distance from the occlusal tip of the crown to the defect is considered to provide a better estimate of the age at the time of insult than is the measured distance from the cemento-enamel

junction (CEJ) to the most occlusal aspect of the defect, but the latter method is more practical because of continual wear of the occlusal surface (Buikstra and Ubelaker, 1994). Thus, we measured the position of horizontal grooves and small pits on the facial crown surface in relation to their position relative to the CEJ, using needle-point sliding calipers accurate to 0.1 mm. Multiple hypoplastic lesions on a tooth were scored separately. We then translated the positions of defects on permanent teeth into a crude estimate of the age at which the stress episode occurred, based on the algorithm developed by P. Walker (personal communication; see Appendix) from the developmental sequence by Massler et al. (1941) of enamel mineralization. Although tooth size variation has been shown to affect the age estimate of an enamel hypoplastic defect, the associated error was shown to be limited to approximately  $\pm 6$  months in an archaeological sample (Hodges and Wilkinson, 1990), and therefore we have not further corrected our gross estimates of age-at-insult. Two thirds of the sample were scored by both authors, and presence and location of defects showed high replicability. The minimum threshold for scoring defects was determined by the most experienced observer (N.C.L.), in order to address methodological concerns raised recently (Danforth et al., 1993; Propst et al., 1994). Due to the small sample sizes, the dental arcades were not partitioned by side or jaw for further analysis.

#### Statistical methods of analysis

We employed bivariate chi-square tests of significance to compare observed and expected defect frequencies for the following independent samples: 1) permanent teeth and deciduous teeth; 2) males and females; and 3) time periods. A one-sample chi-square test of significance compared the relative frequencies of linear grooves and discrete pits among the affected teeth. The probability level for rejection of the null hypothesis of no difference in each comparison was set at  $\alpha = 0.05$ .

#### RESULTS

Table 2 presents the tooth count frequencies of enamel defects in the deciduous and

TABLE 2. Tooth count frequencies of enamel hypoplasia in permanent and deciduous dentitions at Mendes<sup>1</sup>

	LEH	Pits	n	N	%
Permanent teeth					
Incisors	11	0	11	206	5
Canines	40	9	49	153	32
Total	51	9	60	359	17
Deciduous teeth					
Incisors	0	1	4	45	9
Canines	0	1	1	23	4
Molars	0	4	4	46	9
Total	0	6	9	114	8

<sup>1</sup> LEH, linear enamel hypoplasia; n, total number of teeth with enamel defects; N, number of observable teeth; %, [number of affected teeth/number of observable teeth]  $\times 100$ . The result is rounded to the nearest percent.



Fig. 2. Linear enamel hypoplasia of the mandibular canine in an adult male from Mendes.

permanent dentitions. Three types of defects were observed: linear enamel hypoplasia (LEH) (Fig. 2); localized pitting; and a horizontal band of brown discoloration which appears in only one deciduous dentition and which is not discussed further. All defect frequencies reported here should be considered minimum frequencies due to the fact that some crown formation periods are hidden from macroscopic view (Hillson and Bond, 1997). Although the sample sizes are very unequal and results therefore must be interpreted cautiously, the permanent teeth account for 87% of the total number of affected teeth. This prevalence over deciduous teeth is significant at  $\alpha = 0.05$  ( $\chi^2 = 5.86$ ).



TABLE 3. Individual count frequencies of enamel hypoplasia at Mendes<sup>1</sup>

Sex	n	N	%
Males	13	21	62
Females	15	26	58
Unknown	24	41	34
Total	52	88	59

<sup>1</sup> Sex differences are not significant statistically. n, number of affected individuals; N, number of observable individuals; %, [number of affected individuals/number of observable individuals]  $\times$  100. The result is rounded to the nearest percent.

The permanent canines display both furrowed and pitted hypoplasia more frequently than do the permanent incisors, but the canines are the least frequently affected of the three deciduous tooth classes. Only permanent canines display more than one hypoplastic lesion on a tooth, with a mean number of 1.4 lesions per affected tooth. There is also a distinction in the type of hypoplasia: the permanent teeth show both grooves and pits, while only pits appear in the deciduous dentition. Overall, the number of teeth with grooves ( $51/473 = 11\%$ ) is more frequent than is the number of teeth with pits ( $15/473 = 3\%$ ), at a rate of more than three to one (significant at  $\alpha = 0.005$ ;  $\chi^2 = 16.17$ ). Although there are several instances of multiple hypoplastic lesions on a single tooth, there was never an instance where both grooves and pits were present on the same tooth. In only 1 of the 8 individuals with a mixed dentition did both deciduous and permanent teeth exhibit enamel hypoplasia, i.e., pits.

Based on the estimates of the timing of hypoplasia development according to Walker's algorithm, stress episodes leading to LEH occurred most commonly between the ages of approximately 3–5 years, although such episodes were experienced by some individuals as young as 1 year and as old as 6 years. The frequency and location of pits on permanent teeth are similar to those of LEH, but pits first appear early during the development of deciduous teeth. No examples of localized hypoplasia of the primary canine (Lukacs and Walimbe, 1998; Skinner and Hung, 1986, 1989) were noted.

Table 3 presents the individual count frequencies of enamel hypoplasia. Of the 88 individuals, 52 (59%) have one or more teeth exhibiting enamel defects. Four of these

TABLE 4. Individual count prevalence of enamel hypoplasia at Mendes for the Old Kingdom (ca. 3000–2180 BC), First Intermediate (ca. 2180–2040 BC), and Greco-Roman (ca. 332 BC–AD 395) periods<sup>1</sup>

Time period	Sex								
	Male		Female		Unknown		Total		
	n	%	n	%	n	%	n	N	%
Old Kingdom	4	33	4	29	2	20	10	16	63
First Intermediate	1	8	3	21	4	33	8	17	47
Greco-Roman	7	24	5	19	5	19	17	39	44
Total <sup>2</sup>	12	26	12	26	11		35	72	49

<sup>1</sup> The decrease in frequency of affected individuals from the Old Kingdom to the First Intermediate period is statistically significant at the 0.10 level, and approaches significance at the 0.05 level. n, number of affected individuals; %, [number of affected individuals/number of observable individuals]  $\times$  100. The result is rounded to the nearest percent. N, number of observable individuals; not including individuals for whom a time period could not be ascribed.

<sup>2</sup> Total number of individuals for each sex in each time period is not the same as the total numbers of males, females, and unknowns given in Table 4, because only 72 of the 88 observable individuals could be reliably assigned to a time period. The sums of rows and columns may not be identical due to rounding.

individuals displayed multiple lesions on one or more teeth. Sixty-two percent of males and 58% of females displayed enamel hypoplasia, a difference that is not statistically significant ( $\chi^2 = 0.08$ ).

Table 4 presents the individual count frequencies of enamel hypoplasia for the three time periods represented at the site. Of the 88 individuals in the total sample, only 72 could be reliably assigned to one of three cultural periods; the earliest period is least well-represented. The decrease from a 63% defect occurrence rate in the Old Kingdom to 47% in the First Intermediate period is not statistically significant at  $\alpha = 0.05$  ( $\chi^2 = 3.83$ ).

## DISCUSSION

Although hypoplasia in the deciduous teeth from Mendes appears in the earliest forming enamel (i.e., during prenatal development until the first postnatal months), the frequency of these defects is relatively low (8%). This observed frequency is only slightly higher than that for children in modern developed countries and for well-nourished children in less developed countries (usually 5% or less), and is much lower than that considered typical of disadvantaged children in developed and developing nations; in the latter, some 20–70% of children are likely to show hypoplasia of the

primary teeth (Enwonwu, 1973; Infante and Gillespie, 1974; Seow, 1991; Sweeney et al., 1969, 1971). The prevalence of enamel hypoplasia in the permanent teeth at Mendes is similarly consistent with modern rates for well-nourished individuals in good health, although it is significantly higher, statistically, than the deciduous hypoplasia frequency.

The cause of these defects at Mendes is not easy to ascertain, but since inherited conditions are rarely involved in clinical cases and there is no other evidence in this sample for the inherited conditions that lead to enamel defects (e.g., congenital syphilis; see Hillson et al., 1998), one or more acquired conditions is likely responsible.

Acquired causes of enamel defects include systemic and localized factors, the majority of which fall into three categories: trauma, malnutrition, and infection (Seow, 1991; Skinner and Goodman, 1992; Skinner and Hung, 1986, 1989). Trauma is not likely a contributing factor, since the two hypoplastic forms it is known to cause (i.e., LEH of the neonatal line, due to breech presentation or prolonged labor; and localized hypoplasia of the deciduous canine) were not seen in this sample. Alternatively, severe general undernutrition as well as deficiencies of vitamins A and D and of calcium can result in enamel hypoplasia. Prenatally originating defects are often linked to calcium deficiency in the mother, due to malnutrition, malabsorption, or avoidance of dairy products due to lactose intolerance. Postnatally originating defects are often due to hypocalcemia in the infant, which may result from insufficient calcium consumption or from gastrointestinal malabsorption. Severe forms of common childhood respiratory and gastrointestinal infections are the third primary cause of enamel defects, although whether such defects are caused by increased body temperature (i.e., fever), cellular damage by invading pathogens, or malfunctions of major organs is unclear. In contrast to the positive association between LEH and tuberculosis in prehistoric Illinois agriculturalists (Knick, 1981), there is no evidence for chronic infectious disease at Mendes (Lovell, 1992), indicating either a low level of infection or poor survival of

infectious diseases. Since the frequency in this sample of porotic hyperostosis, generally agreed to represent acquired iron deficiency (either dietary or infectious in origin), is also very low (Lovell, 1992, n.d.), an interpretation of generally good health among the inhabitants of Mendes would seem to be supported. The fact that 20 of the 24 affected individuals had only a single hypoplastic lesion suggests that early physiological stresses may have provided some form of immunity against later stresses, although the contrasting view is that individuals with enamel defects may have suffered biological damage to the immune system that resulted in their early death. This latter view has found support in the observation of early mortality among individuals with enamel defects in a large sample of food collectors from the prehistoric Libben site (Duray, 1996). Similarly, although there is no evidence of periodicity in hypoplastic defects that might indicate seasonal or otherwise recurrent stress episodes, stress may have been sufficiently severe as to leave few survivors.

Since LEH and pits occurred in the same individual so rarely (i.e., in only 1 of 24 affected individuals), the two types of defects may have different causative mechanisms, at least in this sample. Although Hillson and Bond (1997) cautioned that assigning an age at time of disruption to pits, in particular, is fraught with difficulty, the age of peak stress in this sample could be associated with weaning, which is often cited as increasing a child's susceptibility to malnutrition and infectious disease (e.g., Fink and Merbs, 1991; Moggi-Cecchi et al., 1994). The human immune system matures at about 2 years of age, and before this time the passive immunity obtained from the mother by the nursing child reduces the risk of ill health. Maternal breast milk is only about 5% protein, however, suggesting that prolonged breast-feeding may lead to protein insufficiency in the face of the high protein and calorie demands of the growing child. By the time of weaning, an increasing amount of liquids and solid food likely was replacing breast milk as nourishment (Katzenberg et al., 1996), but protein in the supplemental foods may have been inad-

equate. One study of infant mortality in ancient Egypt revealed that children aged 3–4 years died more commonly than did those of younger ages, and this pattern was thought to indicate the effects of weaning stresses (Strouhal and Bares, 1993). Breast-feeding in ancient Egypt typically lasted as long as 3 years (Darby et al., 1977, p 56; Lichtheim, 1976, p 141). It is not likely that the age distribution observed here is due to affected children being systematically omitted from this study due to intramural, rather than cemetery, interment, since no previous studies have reported intramural burial at Mendes.

Gastrointestinal tract infection from the consumption of contaminated food and water also may result in substantial levels of childhood morbidity and mortality (Katzenberg et al., 1996) and is a distinct possibility in ancient Egypt, given that medical papyri identify gastrointestinal infection as the most common illness of infancy and prescribe the use of spells and amulets as prophylaxis and treatment for both mother and child (Robins, 1993, pp 85–88). In a study of contemporary weaning practices in a Gambian village, the usual weaning food was a millet gruel that was prepared early in the morning and left standing for the rest of the day; time and fuel were too scarce to permit the preparation of frequent meals. Not only was the gruel nutritionally inadequate, but between one third and one half of the gruel sampled had unacceptable levels of pathogens, including *E. coli* and *Salmonella*, after only 2 hr of standing time (Barrell and Rowland, 1979; cited in Lewis, 1993). Water contaminated with fecal bacteria was considered the likely source of the pathogens and the moist gruel was an excellent medium for bacterial growth.

The higher prevalence of enamel defects among males compared to females at Mendes is not statistically significant, although our results should be viewed cautiously since sex could be determined for only 49% of the individuals in the study sample. These results are consistent with interpretations of ancient Egyptian textual data, however, which indicate that the ancient Egyptians cherished both male and female children (Janssen and Janssen, 1996; Robins, 1993;

Strouhal, 1992; Tyldesley, 1994). Classical authors observed that, in contrast to the Greeks and Romans, Egyptians did not practice infanticide (Lesko, 1996; Tyldesley, 1994). Both men and women in ancient Egypt were legally independent persons with equal rights and the same expectation of a life after death. Both were entitled to inherit wealth and a portion of parental estates.

Although not statistically significant, whether the observed decrease in defect frequency from the Old Kingdom to the First Intermediate period should be considered biologically or culturally meaningful warrants further discussion. Although we cannot reject the null hypothesis that there is no decrease in occurrence rate, this does not prove that the null hypothesis is correct, only that our data do not justify rejecting it. As such, we should consider any additional knowledge that may guide our decision. With a small sample such as this, a test is likely to produce a significant result only if the null hypothesis is very wrong, but the departures from the null hypothesis may be important in substance. Favoring at least tentative acceptance of the observed decrease is the fact that such a pattern is consistent with epigraphic and other data which document prolonged drought and malnutrition during the late Old Kingdom period. The causeway at Saqqara that connects the valley and mortuary temples of Unas (the last pharaoh of the Old Kingdom's Fifth Dynasty) depicts reliefs of emaciated men, women, and children suffering from the effects of severe famine (Fig. 3). Although subject to differing interpretations, the collapse of the Old Kingdom is increasingly considered by scholars to be the end result of a period of climatic aberration that led to a series of low levels of the Nile River, thus discrediting Pharaoh, the god incarnate who controlled the Nile flood, and destroying the Egypt's political and economic systems (e.g., Aldred, 1984; Baines and Málek, 1990; Grimal, 1992; Kemp, 1983; Watterson, 1997). It is believed that famine, poverty, and social upheaval characterized life in Egypt for at least 30 years during the late Old Kingdom and early First Intermediate period. Although the worst droughts occurred in southern Egypt and the hinter-



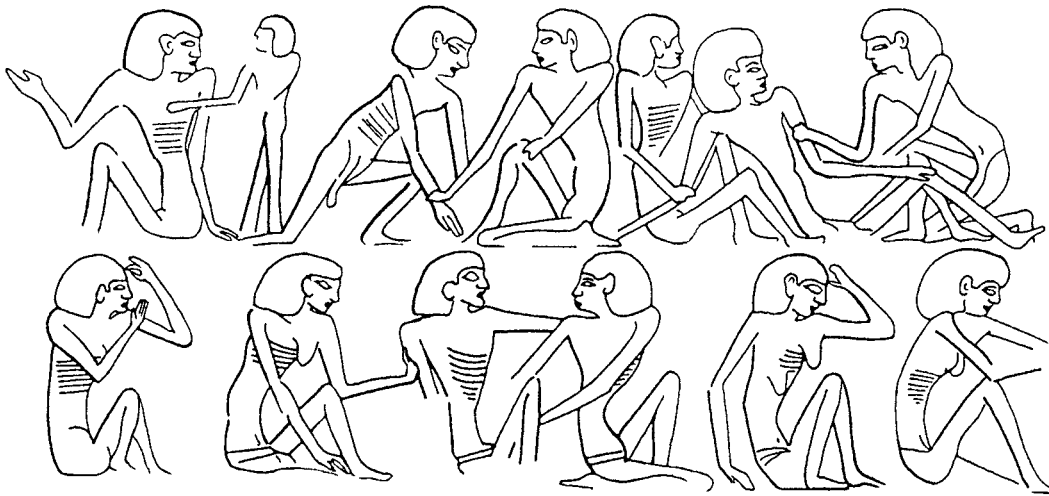


Fig. 3. Depiction of starvation on a carved limestone block from Saqqara (drawn from the original, Egyptian Museum, Cairo).

lands, the delta was not immune from strife, since famine-plagued immigrants from Upper Egypt and from neighboring Libya and Palestine attempted to find respite there. The First Intermediate period eventually, although not without struggle, restored divine power to the king and reestablished the economic health of Egypt via improved irrigation methods and trade networks. By the time of the Greco-Roman period, life in Egypt appears to have settled into a pattern of development in agriculture and commerce, particularly land reclamation activity, which generated wealth for the country as a whole (Bowman, 1989; Ellis, 1992; Grimal, 1992). Periods of economic decline and political strife seem to have been most notable in Upper Egypt, rather than the delta (Watterson, 1997). The latter region's apparent increase in prosperity provides a context that is not inconsistent with the temporal patterning of enamel defects seen at Mendes. While Egypt may have suffered from excessive taxation and lack of autonomy under Roman rule, it is generally agreed that it was not until the late Roman period, with Christianity destroying traditional Egyptian culture, that the populace of Egypt suffered in a fashion similar to that experienced during the demise of the Old Kingdom period.

Thus, it would seem appropriate to examine the relationship between defect frequency and time period further. The chi-square statistic tests whether the observed departure from expectation is more than what random chance would suggest, and its magnitude is a function of sample size: the larger the sample, the greater the chance for significant results. The alpha level deals only with one's willingness to commit a Type I error, i.e., to incorrectly reject a true null hypothesis. The smaller the alpha, the larger the probability of committing a Type II error (i.e., the incorrect acceptance of a false null hypothesis), unless the sample size is increased. Thus, a greater rate of Type I error (e.g.,  $\alpha > 0.05$ ) is often acceptable when the sample is small. With any significance test we are asking if there is a relationship between the variables, and we will have more confidence in our answer if it is based on a large number of observations. In this case, given the context, we have reason to believe that the difference may be biologically and culturally meaningful. Although not significant at the 0.05 level, the calculated  $\chi^2$  in this comparison is significant at  $\alpha = 0.10$ , and thus we suggest that judgment be reserved until additional research is able to confirm or refute a relationship between

the frequency of enamel defects and the time period in which they occur.

### CONCLUSIONS

Our analysis of enamel defects of the deciduous and permanent dentitions from Mendes leads us to the following conclusions:

1. Some 59% of the 88 individuals in the Mendes skeletal sample exhibit enamel defects. The overall frequencies of defects in both the deciduous and permanent teeth are low, however, with tooth count frequencies of 8% and 17%, respectively. These tooth count data are consistent with modern clinical frequencies for well-nourished and healthy children. Since low frequencies of periosteal reactions and porotic hyperostosis also have been observed in the skeletal sample from Mendes, these results support a similar interpretation of maternal, infant, and childhood health at Mendes.
2. The permanent teeth account for 87% of the total number of teeth exhibiting hypoplasia, and show both LEH and localized pits, while deciduous teeth display only pits. These results suggest that different stresses are responsible for enamel hypoplasia in the different age intervals associated with the development of deciduous and permanent teeth. Further, calculation of the age of insult indicates that stress episodes leading to LEH occurred between the ages of approximately 2–6 years, with the peak ages of occurrence between 3–5 years. Stress episodes leading to localized pits, in contrast, may have first occurred in utero. Thus, although a small proportion of the population appears to have experienced physiological stress, these differences are consistent with patterns noted previously in the literature, whereby hypoplasia in the deciduous dentition often reflects maternal and neonatal hypocalcemia, while hypoplasia in the permanent dentition reflects nutritional and infectious stresses that are commonly associated with the process of weaning.
3. Although males exhibited more enamel hypoplasia than did females, the differ-

ence is not statistically significant. These results must be considered tentative, since only half of the skeletal sample could be assigned a sex, but they are in accord with textual accounts of child welfare in ancient Egypt.

4. The pattern of enamel hypoplasia frequencies during the three cultural periods represented by the skeletal sample suggests that episodic stress was more prevalent during the Old Kingdom than during subsequent periods. Although the result is not statistically significant at the 0.05 level, further investigation of temporal patterning in hypoplasia at Mendes and other deltaic sites would seem warranted, given the epigraphic and other evidence for environmental deterioration and consequent malnutrition and poor health in the late Old Kingdom period, evidence that engenders controversy over its documentary vs. metaphorical nature.

Although enamel hypoplasias are not perfect indicators of nutritional status (Goodman and Rose, 1991), the patterns of dental enamel defects among the population at Mendes clearly demonstrate potential for providing information about the health status of ancient Egyptians that complements data obtained from the wealth of documentary sources.

### ACKNOWLEDGMENTS

We thank Bonnie Gustav and Donald Hansen for making available for study the skeletal material excavated in previous decades. More recent excavations at Mendes were completed with the assistance of Marnie Bartell, Ping Lai, and Tracy Prowse in 1992; Margaret Judd in 1996; and Scott Haddow and Sarah Walshaw in 1997. We thank Leslie Dawson for supplying us with a number of useful references and Donald Redford and Robert Wenke for their encouragement and intellectual support of skeletal biology research at Mendes. We are also grateful to the anonymous reviewers who suggested improvements to the paper. This study was supported by Social Sciences and Humanities Research Council of Canada research grants 410-91-1162 and 410-95-0254 to N.L.

APPENDIX. Formulae for calculating age (in years) of formation of hypoplastic lines on permanent teeth (P. Walker, personal communication)<sup>1</sup>

Tooth	Formulae for maxillary teeth	Formula for mandibular teeth
I1	Age = $-0.43939394 \times \text{Ht} + 4.550$	Age = $-0.46551724 \times \text{Ht} + 3.900$
I2	Age = $-0.39655172 \times \text{Ht} + 4.600$	Age = $-0.42187500 \times \text{Ht} + 3.900$
C	Age = $-0.60937500 \times \text{Ht} + 6.000$	Age = $-0.58823529 \times \text{Ht} + 6.500$
P1	Age = $-0.50000000 \times \text{Ht} + 5.900$	Age = $-0.63461538 \times \text{Ht} + 5.850$
P2	Age = $-0.47671492 \times \text{Ht} + 6.061$	Age = $-0.63461538 \times \text{Ht} + 6.850$
M1	Age = $-0.45384615 \times \text{Ht} + 3.570$	Age = $-0.45769231 \times \text{Ht} + 3.560$
M2	Age = $-0.62500000 \times \text{Ht} + 7.500$	Age = $-0.56521739 \times \text{Ht} + 6.950$
M3	Age = $-1.42857143 \times \text{Ht} + 16.00$	Age = $-1.36363636 \times \text{Ht} + 16.00$

<sup>1</sup> I, incisor; C, canine; P, premolar; M, molar; Ht, height, in mm, of the enamel defect from the cemento-enamel junction.

## LITERATURE CITED

- Aldred C. 1984. The Egyptians, revised edition. London: Thames and Hudson. 216 p.
- Baines J, Málek J. 1990. Atlas of ancient Egypt. New York: Facts on File. 240 p.
- Bass W. 1989. Human osteology, 3rd ed. Columbia: Missouri Archaeological Society. 327 p.
- Blakey ML, Armelagos GJ. 1985. Deciduous enamel defects in prehistoric Americans from Dickson Mounds: prenatal and postnatal stress. *Am J Phys Anthropol* 66:371–380.
- Bowman AK. 1989. Egypt after the pharaohs. Berkeley: University of California Press. 268 p.
- Brewer DJ, Wenke RJ. 1992. Transitional late Predynastic-early Dynastic occupations at Mendes: a preliminary report. In: van den Brink EC, editor: The Nile delta in transition. Jerusalem: R. Pinkhaus, p 191–197.
- Buikstra JE, Ubelaker DH. 1994. Standards for data collection from human skeletal remains. Fayetteville, AR. Arkansas Archeological Survey Research Series no. 44. 206 p.
- Condon K, Rose JC. 1992. Intertooth and intratooth variability in the occurrence of developmental enamel defects. *J Paleopathol Monogr Publ* 2:61–77.
- Cook DC, Buikstra JE. 1979. Health and differential survival in prehistoric populations: prenatal dental defects. *Am J Phys Anthropol* 51:649–664.
- Danforth ME, Herndon KS, Propst KB. 1993. A preliminary study of replication in scoring linear enamel hypoplasias. *Int J Osteoarchaeol* 3:297–302.
- Darby WJ, Ghalioungui P, Grivetti L. 1977. Food: The gift of Osiris. London: Academic Press. 877 p.
- Duray SM. 1996. Dental indicators of stress and reduced age at death in prehistoric native Americans. *Am J Phys Anthropol* 99:275–286.
- Ellis SP. 1992. Graeco-Roman Egypt. Aylesbury, UK: Shire Egyptology. 56 p.
- Enwonwu CO. 1973. Influence of socio-economic conditions on dental development in Nigerian children. *Arch Oral Biol* 18:95–108.
- Fink TM, Merbs CF. 1991. Paleonutrition and paleopathology of the Salt River Hohokam: a search for correlates. *Kiva* 56:293–317.
- Goodman AH, Armelagos GJ. 1985. Factors affecting the distribution of enamel hypoplasia within the human permanent dentition. *Am J Phys Anthropol* 68:479–493.
- Goodman AH, Rose JC. 1990. Assessment of systemic physiological perturbations from dental enamel hypoplasias and associated histological structures. *Yrbk Phys Anthropol* 33:59–110.
- Goodman AH, Rose JC. 1991. Dental enamel hypoplasias as indicators of nutritional status. In: Kelley MA, Larsen CS, editors. Advances in dental anthropology. New York: Wiley-Liss. p 279–293.
- Goodman AH, Armelagos GJ, Rose JC. 1980. Enamel hypoplasias as indicators of stress in three prehistoric populations from Illinois. *Hum Biol* 52:515–528.
- Grimal N. 1992. A history of ancient Egypt. Oxford: Blackwell. 512 p.
- Hansen DP. 1967. Mendes 1965 and 1966. I. The excavations at Tell el Rub'a. *J Am Res Center Egypt* 6:5–16.
- Hillson S. 1979. Diet and dental disease. *World Archaeol* 11:147–162.
- Hillson S. 1996. Dental anthropology. Cambridge: Cambridge University Press. 373 p.
- Hillson S, Bond S. 1997. Relationship of enamel hypoplasia to the pattern of tooth crown growth: a discussion. *Am J Phys Anthropol* 104:89–103.
- Hillson S, Grigson C, Bond S. 1998. Dental defects of congenital syphilis. *Am J Phys Anthropol* 107:25–40.
- Hodges DC, Wilkinson RG. 1990. Effect of tooth size on the aging and chronological distribution of enamel hypoplastic defects. *Am J Hum Biol* 2:553–560.
- Holtz RK, Steiglitz D, Hansen DP, Ochsenschlager E. 1980. Mendes I. Cairo: American Research Center in Egypt. 83 p.
- Infante PF, Gillespie GM. 1974. An epidemiologic study of linear enamel hypoplasia of deciduous anterior teeth in Guatemalan children. *Arch Oral Biol* 19:1055–1061.
- Janssen RM, Janssen JJ. 1996. Growing up in ancient Egypt. London: Rubicon. 192 p.
- Katzenberg MA, Herring DA, Saunders SR. 1996. Weaning and infant mortality: evaluating the skeletal evidence. *Yrbk Phys Anthropol* 39:177–199.
- Kemp BJ. 1983. Old Kingdom, Middle Kingdom and Second Intermediate period c. 2686–1552 BC. In: Trigger BG, Kemp BJ, O'Connor D, Lloyd AB, editors. Ancient Egypt, a social history. Cambridge: Cambridge University Press. p 71–182.
- Knick SG III. 1981. Linear enamel hypoplasia and tuberculosis in pre-Columbian North America. *Ossa* 8:131–138.
- Lesko BS. 1996. The remarkable women of ancient Egypt, 3rd ed. Providence: B.C. Scribe Publications. 68 p.
- Lewis G. 1993. Some studies of social causes of and cultural response to disease. In: Mascie-Taylor CGN, editor. The anthropology of disease. Oxford: Oxford University Press, p 73.
- Lichtheim M. 1976. The instruction of Any. Ancient Egyptian literature volume II: the New Kingdom. Berkeley: University of California Press. p 135–146.
- Lovell NC. 1992. The 1992 excavations at Kom el-Adhem, Mendes. *J Soc Stud Egypt Antiq* 21/22:21–36.

- Lukacs JR. 1992. Dental paleopathology and agricultural intensification in South Asia: new evidence from Bronze Age Harappa. *Am J Phys Anthropol* 87:133–150.
- Lukacs JR. 1994. The osteological paradox and the Indus civilization: problems inferring health from humans skeletons at Harappa. In: Kenoyer JM, editor. *From Sumer to Meluhha*. Wisconsin Archaeological Reports, volume 3. Madison: University of Wisconsin. p 143–155.
- Lukacs JR, Walimbe SR. 1998. Physiological stress in prehistoric India: new data on localized hypoplasia of primary canines linked to climate and subsistence change. *J Archaeol Sci* 25:571–585.
- Magee MJ, Wayman ML, Lovell NC. 1996. Chemical and archaeological evidence for the destruction of a sacred animal necropolis at ancient Mendes, Egypt. *J Archaeol Sci* 23:485–492.
- Massler M, Schour I, Poncher H. 1941. Developmental pattern of the child as reflected in the calcification pattern of the teeth. *Am J Dis Child* 62:33–67.
- May RL, Goodman AH, Meindl RS. 1993. Response of bone and enamel formation to nutritional supplementation and morbidity among malnourished Guatemalan children. *Am J Phys Anthropol* 92:37–51.
- McKeown AH, Bennett JL. 1995. A preliminary investigation of postmortem tooth loss. *J Forensic Sci* 40:755–757.
- Moggi-Cecchi J, Pacciani E, Pinto-Cisternas J. 1994. Enamel hypoplasia and age at weaning in 19th century Florence, Italy. *Am J Phys Anthropol* 93:299–306.
- Nikiforuk G, Fraser D. 1981. The etiology of enamel hypoplasia: a unifying concept. *J Pediatr* 98:888–893.
- Pindborg JJ. 1970. Pathology of the dental hard tissues. Philadelphia: W.B. Saunders. 442 p.
- Propst KB, Danforth ME, Jacobi J. 1994. Replicability in scoring enamel hypoplasias: a preliminary report. *Paleopathol News* 87:11–12.
- Redford DB. 1993a. ATP excavations at Mendes. *Soc Stud Egypt Antiq News* Sept: 1.
- Redford DB. 1993b. The 1993 summer expedition to Mendes. *Akhenaton Temple Project News* 3:1–3.
- Redford DB, Mumford GD, Redford S, Schubert SB, Hummel R. 1988. The first season of excavations at Mendes (1991). *J Soc Stud Egypt Antiq* 18:49–79.
- Robins G. 1993. *Women in ancient Egypt*. Cambridge: Harvard University Press. 205 p.
- Rose JC, Condon K, Goodman AH. 1985. Diet and dentition: developmental disturbances. In: Gilbert R, Mielke J, editors. *The reconstruction of prehistoric diets*. New York: Academic Press, p 281–305.
- Sarnat B, Schour I. 1941. Enamel hypoplasia (chronological enamel aplasia) in relation to systemic disease: a chronologic, morphologic and etiologic classification. *J Am Dent Assoc* 29:67–75.
- Seow WK. 1991. Enamel hypoplasia in the primary dentition: a review. *J Dent Child* 58:441–452.
- Skinner MF, Goodman AH. 1992. Anthropological uses of developmental defects of enamel. In: Saunders SR, Katzenberg MA, editors. *Skeletal biology of past peoples: research methods*. New York: Wiley-Liss. p 153–174.
- Skinner MF, Hung JTW. 1986. Localized enamel hypoplasia of the primary canine. *J Dent Child* 53:197–200.
- Skinner MF, Hung JTW. 1989. Social and biological correlates of localized enamel hypoplasia of the human deciduous canine tooth. *Am J Phys Anthropol* 79:159–175.
- Strouhal E. 1992. *Life of the ancient Egyptians*. Norman: University of Oklahoma Press. 279 p.
- Strouhal E, Bares L. 1993. *Secondary cemetery in the Mastaba of Ptahshepses at Abusir*. Prague: Charles University.
- Sweeney EA, Camberra J, Mata Z. 1969. Factors associated with linear hypoplasia of human deciduous teeth. *J Dent Res* 48:1275–1279.
- Sweeney EA, Saffir AJ, de Leon R. 1971. Linear hypoplasia of deciduous teeth in malnourished children. *Am J Clin Dent* 24:29–31.
- Tyldesley J. 1994. *Daughters of Isis*. London: Viking Penguin. 318 p.
- Watterson B. 1997. *The Egyptians*. Cambridge: Blackwell. 347 p.
- Wenke RJ. 1991. The evolution of early Egyptian civilization: issues and evidence. *J World Prehist* 5:279–329.
- White TD. 1991. *Human osteology*. San Diego: Academic Press. 455 p.
- Wilson K. 1982. *Cities of the delta, part II: Mendes*. Preliminary report on the 1979 and 1980 seasons. Malibu: Undena. 43 p.
- Wood JR, Milner GR, Harpending HC, Weiss KM. 1992. The osteological paradox: problems inferring health from skeletal samples. *Curr Anthropol* 33:343–370.